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Irradiation with Water-Filtered Infrared A plus Visible Light Improves Cutaneous Scleroderma Lesions in a Series of Cases

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Key Words
Scleroderma · Water-filtered infrared A · Durometry · Morphea

Abstract
Background: Cutaneous scleroderma is a chronic inflammatory disease of the dermal and subcutaneous connective tissue leading to sclerosis. Sclerosis of the skin can lead to dysmorphism, contractures and restrictions of movement. ***Objective:*** The purpose of the study was to evaluate sclerosis in cutaneous scleroderma patients and to determine the efficacy of water-filtered infrared A plus visible light treatment, wIRA(+VIS), in 10 patients. ***Methods:*** Hardness of the normal and diseased skin was measured by durometry in 10 controls and 8 patients. Moreover, circumscribed scleroderma (CS) was treated with wIRA(+VIS) irradiations in 10 patients who had not responded to conventional therapies. ***Results:*** wIRA(+VIS) therapy led to a marked improvement, persistent even during long-term follow-up, in 7 out of 10 patients with CS. Of the other patients, 1 showed decreased sclerosis and disease activity and developed a worsening after cessation of therapy. In 2 further patients, where previous UVA1 treatment had failed to reduce disease activity, wIRA(+VIS) produced a slight decrease in sclerosis, but disease activity was still present. ***Conclusion:*** wIRA(+VIS) appears to be effective in the treatment of CS. Durometry proved to be helpful in assessing the degree of sclerosis and in documenting the response to therapy in these patients.

Introduction

Localized scleroderma/morphea, linear circumscribed scleroderma (LCS) and generalized circumscribed scleroderma (GCS) comprise the group of cutaneous forms of circumscribed scleroderma (CS), a chronic inflammatory disease of the dermal and subcutaneous connective tissue leading to sclerosis [1]. Internal organs are not involved, and neither Raynaud's phenomenon nor acrosclerosis are present. Morphea is characterized by circumscribed sclerotic plaques in 1 or – at the most – 2 circumscribed areas [1]. In GCS, 3 or more anatomical localizations are affected [1]. LCS appears with a linear distribution and can involve adjacent muscles and bones. Different etiological factors have been reported such as trauma, radiation therapy, vaccination, viral infections including measles and varicella, and bacterial infections such as *Borrelia burgdorferi* [2]. An autoimmune mecha-

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nism is likely [1, 2], characterized in the early phase by an immune activation with inflammatory infiltrates leading to an increased expression of cytokines [3]. This leads to an imbalance of collagen synthesis and degradation by fibroblasts, resulting in sclerosis [1]. Sclerosis of the skin causes pain, restrictions of movement and contractures. Moreover, many patients suffer from dysmorphisms. CS is a rare disease with an age- and sex-adjusted incidence of 2.7/100,000 [2]. In limited forms of CS, a regression of disease activity was found in 50% of cases after 2.5 years [1]. Prognosis is worse for GCS and LCS [1]. In these conditions, sclerotic tissue hardening and skin atrophy often persist. Therapies that stop disease activity and prevent sclerosis are therefore important.

Large, prospective randomized studies on this condition are very rare due to the low number of affected patients. In the case series and case reports published so far, the therapeutic efficacy of topical, intralesional and systemic corticosteroids, topical calcipotriol, topical tacrolimus, imiquimod, intralesional γ-interferon, ultraviolet light A1 (UVA1), psoralen plus UVA (PUVA), topical photodynamic therapy (PDT), oral calcitriol, D-penicillamine, methotrexate, penicillins, sulfasalazine, diphenylhydantoin, clofidine hydrochloride, retinoids and physiotherapy have been described [1, 2]. Interestingly, only treatment regimens including cyclophosphamide, UVA1 or methotrexate have led to a significant reduction in skin sclerosis thus far [3–5]. Wienert et al. [6] and Foerster et al. [7] described infrared A (IRA) to be therapeutically effective in the treatment of scleroderma-associated Raynaud's phenomenon and suggested that IRA could also be helpful in other forms of cutaneous scleroderma. Geissler and Schumann described a case of an ulcerative morphea of the lower leg which was successfully treated with water-filtered IRA plus visible light [wIRA(+VIS)] [8].

The observations reported here indicate that the local irradiation with wIRA(+VIS) may also lead to a marked improvement of CS.

Methods

wIRA(+VIS) Treatment

Over a period of 7.5 years, 10 patients suffering from recalcitrant CS (9 females, 1 male, aged 6–62 years, duration of disease prior to the inclusion in this study between 1 and 7 years) were treated at the Departments of Dermatology, University Hospital RWTH Aachen, Germany, and Inselspital, University Hospital, Bern, Switzerland, using an IR irradiation source emitting wIRA (75%) plus VIS (25%; Hydrosun 500 and 501 with an OG590 filter, Hydrosun Medizintechnik GmbH, Müllheim, Germany). The spectrum of wIRA(+VIS) is emitted by a halogen lamp and filtered by a water-containing cuvette. The spectrum ranges from 580 to 1,400 nm (VIS: 580–780 nm, wIRA: 780–1,400 nm; fig. 1). The distance of the radiator to the skin was 25–28 cm with a total irradiance of 180–200 mW/cm^2 (135–150 mW/cm^2 wIRA, 45–50 mW/cm^2 VIS, Hydrosun 501). The irradiation time was 20–30 min. The radiator is an established form of treatment for several other conditions, such as acute and chronic wounds, warts or PDT of actinic keratoses, and is also used in neonatology and physiotherapy [9–13].

In all patients the CS lesions still showed progressive sclerosis at the time of the initiation of the wIRA(+VIS) treatment. All patients had received several conventional therapies including systemic antibiotics, topical corticosteroids or topical tacrolimus and physiotherapy without success prior to the observational study (table 1). In 2 of them, additional treatment attempts with UVA1 and in another patient local PUVA therapy had also been ineffective (table 1). Therapy with wIRA(+VIS) was attempted before immunosuppressive systemic medication was considered. In all patients wIRA(+VIS) was applied to all plaques 2–5 times per week. Initial clinical assessment included complete physical examination. Standardized photographic documentation was performed before and after therapy. Each patient was examined by the same physicians (V.v.F., K.K.L.) before and once weekly during treatment. Follow-up was carried out over a period of 1–7.5 years after wIRA(+VIS) treatments at the two University hospitals. All patients were monitored clinically (table 1). V.v.F. (Department of Dermatology, University Hospital Aachen) documented the efficacy using a 4-tiered scale (marked improvement, mild to moderate improvement, no improvement, worsening), a visual analog scale (VAS) ranging from 0 = none to 10 = severe, evaluating the overall disease activity of the plaques and taking into account the size, skin texture and hardness as well as color, and in 3 patients by durometry. K.K.L. (Department of Dermatology, University Hospital Berne) used the 4-tiered scale to document the efficacy.

In addition, after evaluation of the durometer technique in healthy controls and scleroderma patients, the sclerosis of 3 patients with CS was followed up using durometry during wIRA(+VIS) treatment.

Durometry

From 2008 to 2010, durometry was used to evaluate the hardness of the normal and sclerotic skin in 8 patients with scleroderma spectrum disorder (CS n = 3, systemic sclerosis n = 5) and 10 healthy controls at the Department of Dermatology, RWTH Aachen University Hospital. Since this method helps to elucidate the differences between diseased and normal skin, data were assessed and reported briefly.

A durometer which had been specifically designed for this purpose was employed (fig. 2, Heinrich Bareiss Prüfgeräte GmbH, Oberdischingen, Germany). The calibrated handset consists of a cylinder with a base. Within the cylinder, a spring is connected to a floating ball which sticks out of the device. A display provides digital readings (0.0–100.0 arbitrary units; fig. 2). For measurements, the device is positioned vertically on the area of interest; the contact pressure is determined taking advantage of the weight of the device. This assessment is fast, noninvasive, painless and lacks side effects. To evaluate the usefulness of the device, skin areas on the back of the right hand, the inside of the forearm, the upper arm, the abdomen and the thigh of 10 healthy volunteers (5 males and 5 females, 6–62 years, normal weight) underwent mea-

Table 1. Patient characteristics

No.	Sex	Age, years	Type of scleroderma	Localization	Pretreatments and comments	wIRA(+VIS) treatments per week	Number of wIRA(+VIS) treatments	Follow-up time after completion of wIRA(+VIS), years	Measurement of sclerosis	Efficacy of wIRA(+VIS) treatments
1	f	6	LCS	Left arm	Topical corticosteroids, restriction of movement of the left arm	2–3	Forearm: 16; upper arm: 22	5	Palpation/ VAS	++ [22]
2	f	15	GCS	Throat, thorax, face	UVA1, topical corticosteroids, tacrolimus, ANA 1:1,240 [2003, before wIRA(+VIS) treatment]	2–3	18	7.5; in 2010 SLE was diagnosed	Palpation/ VAS	+/0
3	m	55	Morphea	Left flank	Topical corticosteroids, itching	2–3	28	2.5	Palpation/ VAS	++
4	f	62	Morphea	Left inguinal	Topical corticosteroids, paresthesia due to irritation of medial femoral cutaneous nerve by sclerosis	2–3	19	2.5	Palpation/ VAS	++
5	f	16	LCS	Right upper arm	Doxycycline, intravenous penicillin, topical corticosteroids	2–3	24	2	Palpation	++
6	f	16	LCS	Left thigh	Doxycycline, topical corticosteroids	2–3	20	5	Palpation	++
7	f	21	LCS	Pelvis, left leg	Doxycycline, intravenous penicillin, intravenous ceftriaxone	2–3	48	1	Palpation	++
8	f	56	GCS	Right forearm, abdomen	Topical corticosteroids, PUVA, tacrolimus, tendinitis and restriction of movement	2–5	26	1	Palpation/ durometry	+
9	f	38	Morphea	Left lower leg	Topical corticosteroids, tacrolimus, pain and discomfort	2–5	19	1	Palpation/ durometry	++
10	f	33	GCS	Abdomen, flank	UVA1, topical corticosteroids, tacrolimus	2–5	25	1	Palpation/ durometry	+/0

Efficacy score: ++ = marked improvement; + = mild to moderate improvement; 0 = no improvement; – = worsening. VAS = Visual analog scale; ANA = antinuclear antibodies; SLE = systemic lupus erythematosus.

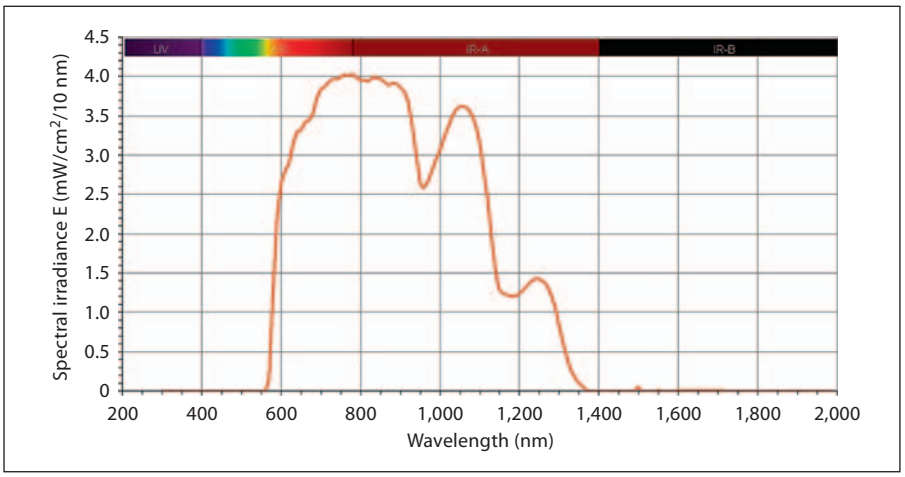


Fig. 1. Spectral irradiance of a wIRA(+VIS) radiator (Hydrosun® 501 with water-containing cuvette and orange filter OG590) at approximately 210 mW/cm^2 total irradiance [11].

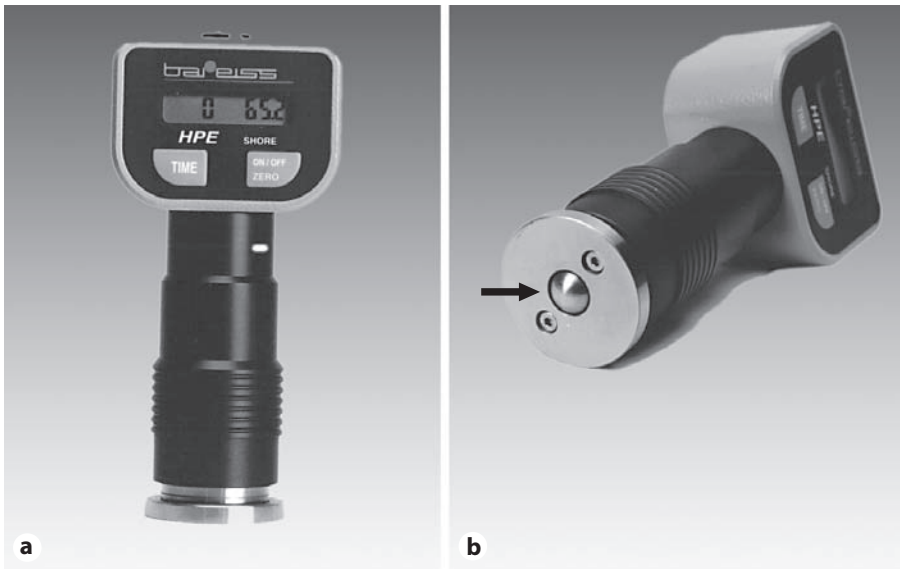


Fig. 2. Durometer with display showing digital readings (**a**) and floating ball (arrow) for measurements (**b**).

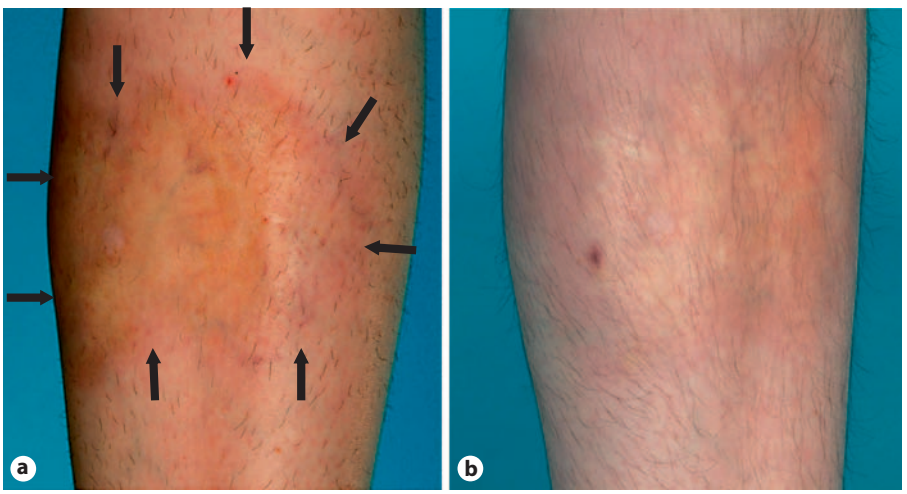


Fig. 3. a CS (arrows) of the left lower leg with sclerosis and hair loss for 5 years (patient No. 9). **b** Softening of the skin, remission of sclerosis, reduction of hyperpigmentation and hair regrowth after wIRA(+VIS) treatment.

surement 5 times within 1 day. To examine reproducibility, the same forearm was evaluated 5 times per day for 5 days. Since hardness is altered by muscle tone, for example of the forearm, all measurements were conducted in a relaxed posture. In addition, no measurements were performed on skin covering bone, since this also affects the hardness. For comparison, the hardness of a scrubbing pad (Optiwish Spontex) and of the tabletop of a hospital desk was assessed by durometry. Finally, the hardness of the sclerotic skin was determined in plaques of 8 scleroderma patients. The durometer was examined and received a works calibration certificate after completion of the investigations (Heinrich Bareiss Prüfgeräte).

Statistical Analysis

Descriptive analysis was performed with median, 25th and 75th percentiles (interquartile range), minimum and maximum (box-and-whisker graphs).

Results

Therapy Efficacy of wIRA(+VIS)

Therapeutic efficacy was assessed descriptively by the physician (marked improvement, mild to moderate improvement, no improvement, worsening; table 1). The number of irradiations ranged from 16 to 48 (table 1). The duration of treatment was based on the therapeutic outcome. In the 2 patients showing an insufficient response to wIRA(+VIS) therapy, the irradiations were most probably terminated too soon since a continuous improvement of skin sclerosis during the treatment period was observed, with rapid deterioration thereafter. A longer irradiation period might have prevented this deterioration. However, because the disease in these 2 young girls was very active, the decision was made not to delay a systemic therapy.

Six patients reported a reduction of pruritus and discomfort of the CS lesion within 4 weeks after initiation of wIRA(+VIS) treatment. In addition, a reduction of the erythema and of the sclerosis was noted in 3 out of 4 patients documented by VAS 0–10 (mean initial VAS = 9, after 4 weeks VAS = 6). Interestingly, patient No. 4, suffering from irritation of the medial femoral cutaneous nerve due to CS, reported steady improvement of paresthesia after 4 weeks of treatment. Within 12 weeks, the hardness of her lesions had completely disappeared (VAS 0–10: baseline = 9, after 12 weeks = 1). The skin felt soft, but hair follicles were still missing and the epidermis remained atrophic. In another female patient (No. 9) reduced sclerosis and skin hardness was detected (durometry 0.0–100.0: baseline = 43.8, after 19 treatments = 17.4–20.4), and hair regrowth was observed (fig. 3).

Five patients, including those with a hyperpigmentation prior to wIRA(+VIS) treatments, showed a slight hyperpigmentation at least at 1 observation point and after the end of treatment (fig. 4c). It is known that CS can heal spontaneously, leaving focal alterations of pigmentation [1]. Following the clinical course after wIRA(+VIS) irradiation a decrease in hyperpigmentation was observed (fig. 3, 4). Moreover, hyperpigmentations or other pigmentary disorders have never been observed thus far after wIRA(+VIS) irradiations for other indications.

The patients reported no other remaining symptoms or disturbances. In all cases the treatment was well tolerated without any other adverse events, and patient compliance was excellent.

Taken together, a marked improvement of CS in 7 of 10 patients was observed (fig. 3, 4). Since during the follow-up of 1–7.5 years after wIRA(+VIS) treatments none of these successfully treated patients experienced worsening, they can be considered cured. In 1 of 10 patients a clinical improvement and decrease in sclerosis were observed. This patient showed subsequent deterioration after cessation of irradiation (fig. 5), possibly because the treatment had been stopped too soon. In the 2 remaining patients who suffered from GCS, UVA1 had failed to inhibit disease progression previously. Limited frequency and duration of wIRA(+VIS) irradiation led to a slight decrease in sclerosis as long as it was used, although disease activity was not altered relevantly. Daily and longer treatment times may lead to more benefit.

From these preliminary data, we conclude that wIRA(+VIS) appears to be effective in the reduction of sclerosis and in achieving disease control in patients with CS.

Durometry

Twenty-five durometer measurements (5 per day on 5 consecutive days) on the same point of skin yielded very similar results, showing the stability of the method (table 2). To demonstrate the hardness of various materials, a tabletop was compared to a scrubbing pad. The tabletop was very hard (median: 99.2) compared to the scrubbing pad (median: 26.6). Moreover, measurements were performed on the back of the right hand, the inside of the forearm, the upper arm, the abdomen and the thigh of 10 healthy volunteers and 8 patients (CS n = 3, systemic sclerosis n = 5). Compared to the healthy controls, the affected skin of patients was clearly harder, irrespective of the site examined (fig. 6a–d). Nonafflicted skin of patients showed durometer values similar to those obtained for the skin of healthy controls. The skin of the healthy

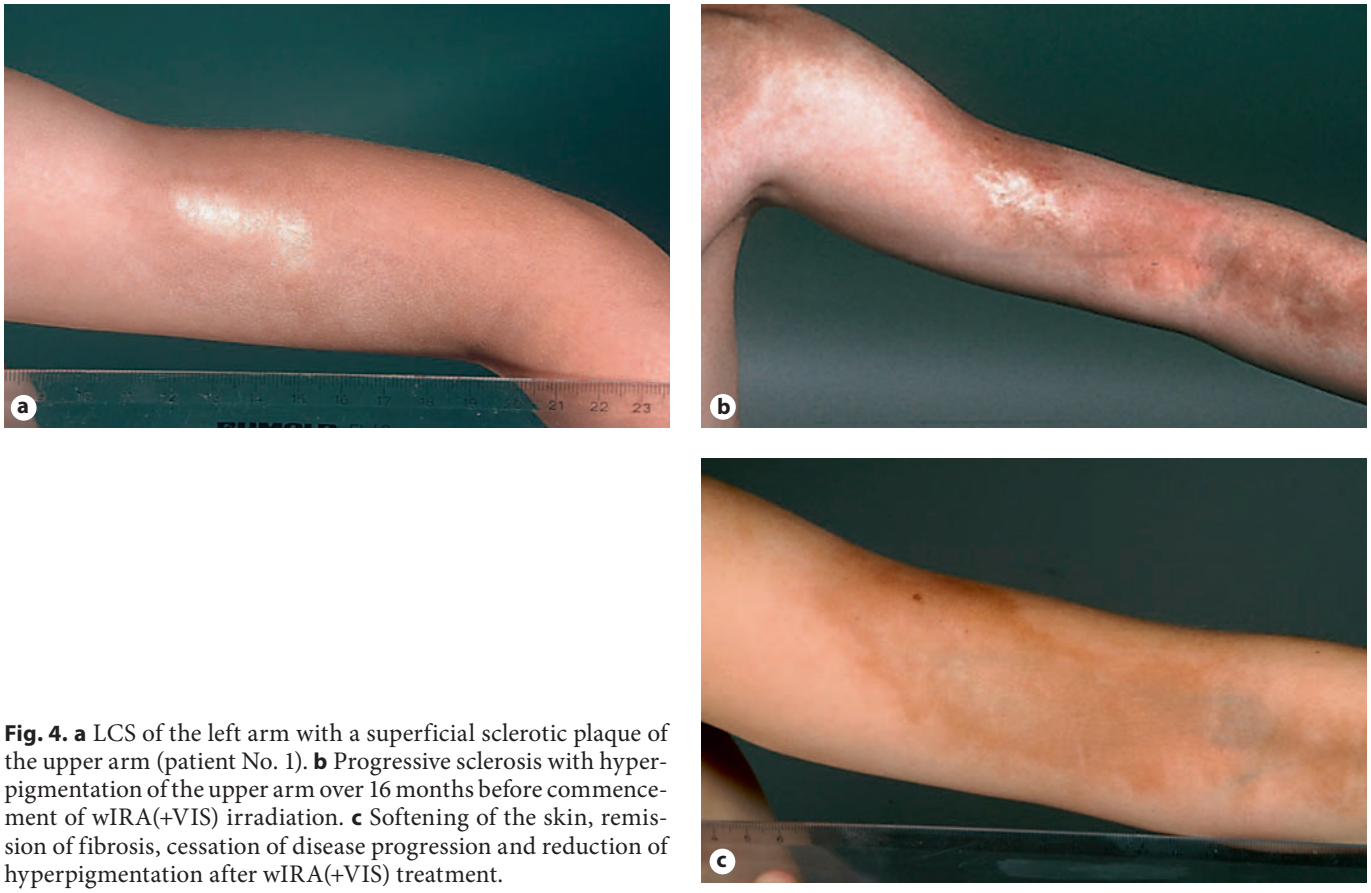


Fig. 4. a LCS of the left arm with a superficial sclerotic plaque of the upper arm (patient No. 1). **b** Progressive sclerosis with hyperpigmentation of the upper arm over 16 months before commencement of wIRA(+VIS) irradiation. **c** Softening of the skin, remission of fibrosis, cessation of disease progression and reduction of hyperpigmentation after wIRA(+VIS) treatment.

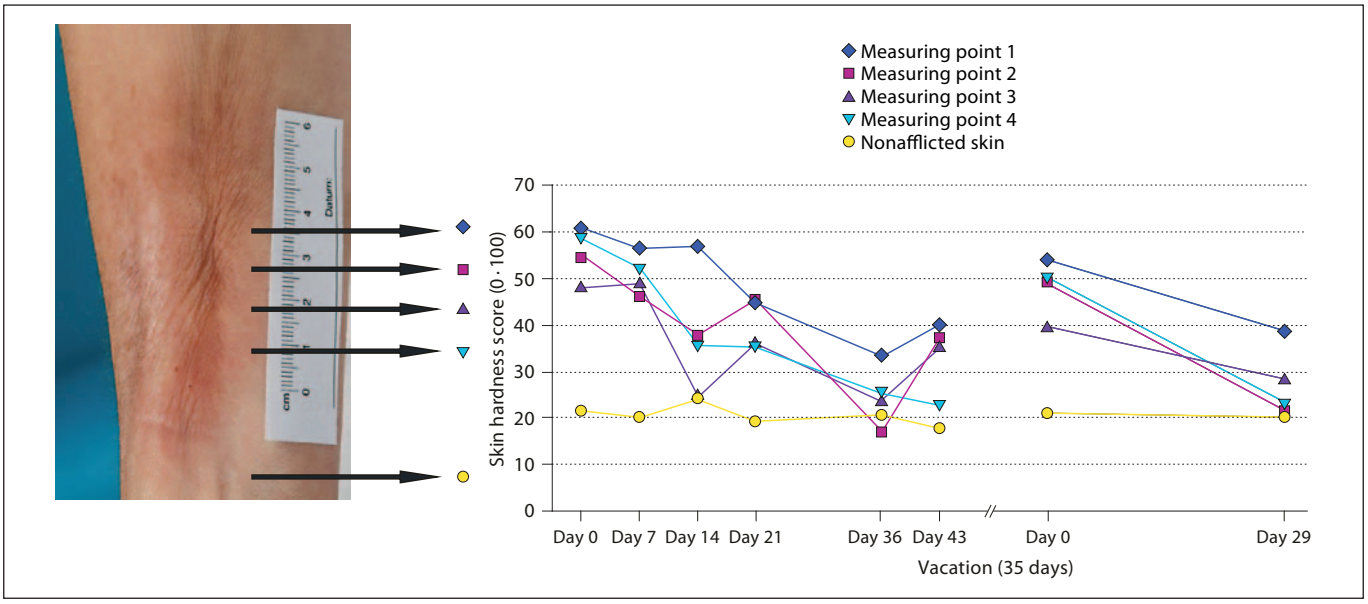


Fig. 5. Changes in skin hardness during wIRA(+VIS) therapy measured by durometry (0.0–100.0) of 4 measuring points of 1 sclerotic plaque of the forearm in a single patient (No. 8), examined at 8 different time points. After 43 days, the patient stopped treatment (vacation) and worsening developed within 5 weeks. wIRA(+VIS) was started again, resulting in improvement. The measured values also demonstrate the heterogeneity within a given lesion.

Table 2. Durometry results obtained from the same site of the right forearm of 10 healthy volunteers under standardized conditions (5 consecutive measurements per day, on 5 consecutive days, presented in the table sorted by rank) on a scale of 0.0–100.0

	Day 1	Day 2	Day 3	Day 4	Day 5	Median of the medians of days 1–5	Median of the ranges 'max.-min.' of days 1–5
Volunteer 1							
Maximum	19.9	25.4	21.0	21.2	19.7		
	19.8	24.4	16.6	20.2	16.6		
Median	19.4	24.3	16.1	20.1	16.1	19.4	
	19.2	23.7	15.6	20.0	15.6		
Minimum	18.7	22.0	13.9	18.6	15.1		
Max.-min.	1.2	3.4	7.1	2.6	4.6		3.4
Volunteer 2							
Maximum	19.9	17.6	21.7	23.4	19.0		
	19.8	15.7	20.8	22.2	19.0		
Median	18.1	14.6	20.2	22.0	18.5	18.5	
	18.0	14.4	19.4	20.4	16.8		
Minimum	17.7	14.2	18.9	20.3	16.2		
Max.-min.	2.2	3.4	2.8	3.1	2.8		2.8
Volunteer 3							
Maximum	18.7	14.2	12.6	12.0	16.8		
	18.0	14.1	9.6	11.7	12.5		
Median	16.7	13.4	9.1	10.1	12.0	12.0	
	16.7	13.2	8.8	9.8	11.5		
Minimum	16.5	11.4	8.4	9.8	10.6		
Max.-min.	2.2	2.8	4.2	2.2	6.2		2.8
Volunteer 4							
Maximum	25.5	17.6	23.0	21.9	18.2		
	24.6	17.2	22.2	19.7	18.1		
Median	23.1	16.6	22.0	19.2	18.0	19.2	
	23.0	16.5	21.6	19.1	17.2		
Minimum	22.5	16.2	21.6	18.8	16.7		
Max.-min.	3.0	1.4	1.4	3.1	1.5		1.5
Volunteer 5							
Maximum	14.0	15.2	12.9	15.8	15.7		
	11.8	15.2	12.0	14.0	13.9		
Median	11.7	14.2	11.0	13.2	12.6	12.6	
	11.4	12.2	11.0	12.7	11.7		
Minimum	11.0	10.9	10.5	12.1	9.9		
Max.-min.	3.0	4.3	2.4	3.7	5.8		3.7
Volunteer 6							
Maximum	22.1	29.0	31.4	32.7	26.2		
	20.5	23.7	28.3	31.1	25.2		
Median	20.4	23.0	28.1	30.4	23.4	23.4	
	19.9	22.6	27.6	29.2	22.2		
Minimum	18.6	21.3	26.0	28.9	20.6		
Max.-min.	3.5	7.7	5.4	3.8	5.6		5.4
Volunteer 7							
Maximum	21.6	18.7	17.8	14.2	16.1		
	19.7	18.6	17.0	14.1	15.7		
Median	19.1	18.5	16.2	13.8	15.5	16.2	
	18.4	18.1	15.7	13.5	15.2		
Minimum	18.0	17.6	15.5	12.9	14.6		
Max.-min.	3.6	1.1	2.3	1.3	1.5		1.5

Table 2 (continued)

	Day 1	Day 2	Day 3	Day 4	Day 5	Median of the medians of days 1–5	Median of the ranges 'max.-min.' of days 1–5
Volunteer 8							
Maximum	20.6	23.6	19.0	18.2	21.9		
	20.3	23.2	18.1	17.2	21.8		
Median	19.6	23.0	17.8	16.8	21.2	19.6	
	19.4	22.8	17.0	16.6	20.8		
Minimum	18.7	21.9	11.4	14.9	17.2		
Max.-min.	1.9	1.7	7.6	3.3	4.7		3.3
Volunteer 9							
Maximum	17.3	18.6	16.0	19.4	23.3		
	16.8	18.0	15.5	18.4	22.1		
Median	15.7	16.0	15.4	18.1	20.1	16.0	
	13.8	15.7	14.8	17.9	18.4		
Minimum	13.7	15.4	13.0	17.6	17.5		
Max.-min.	3.6	3.2	3.0	1.8	5.8		3.2
Volunteer 10							
Maximum	22.7	21.0	23.4	24.8	19.8		
	22.0	21.0	23.0	22.1	19.7		
Median	22.0	20.9	22.0	22.0	19.0	22.0	
	21.8	20.7	21.5	21.1	18.6		
Minimum	20.9	18.6	21.1	20.8	18.3		
Max.-min.	1.8	2.4	2.3	4.0	1.5		2.3
Median of the medians of volunteers 1–10	19.3	17.6	17.0	18.7	18.3		

The 250 readings of the healthy volunteers were in the relatively small range of 8.4–32.7 on a scale of 0.0–100.0 whereas hardness of plaques of the forearm ranged up to 65.0. In addition, the measurements were stable for each individual volunteer both within each day and within the 5-day observation period. Thus, clear differentiation even within the group of healthy volunteers was possible. For example, all 25 measurements of volunteer 5 were lower than the lowest value of the 25 measurements of volunteer 6 and lower than the lowest value of the 25 measurements of volunteer 4. These disjunct values even within the group of healthy persons indicate softer versus harder skin.

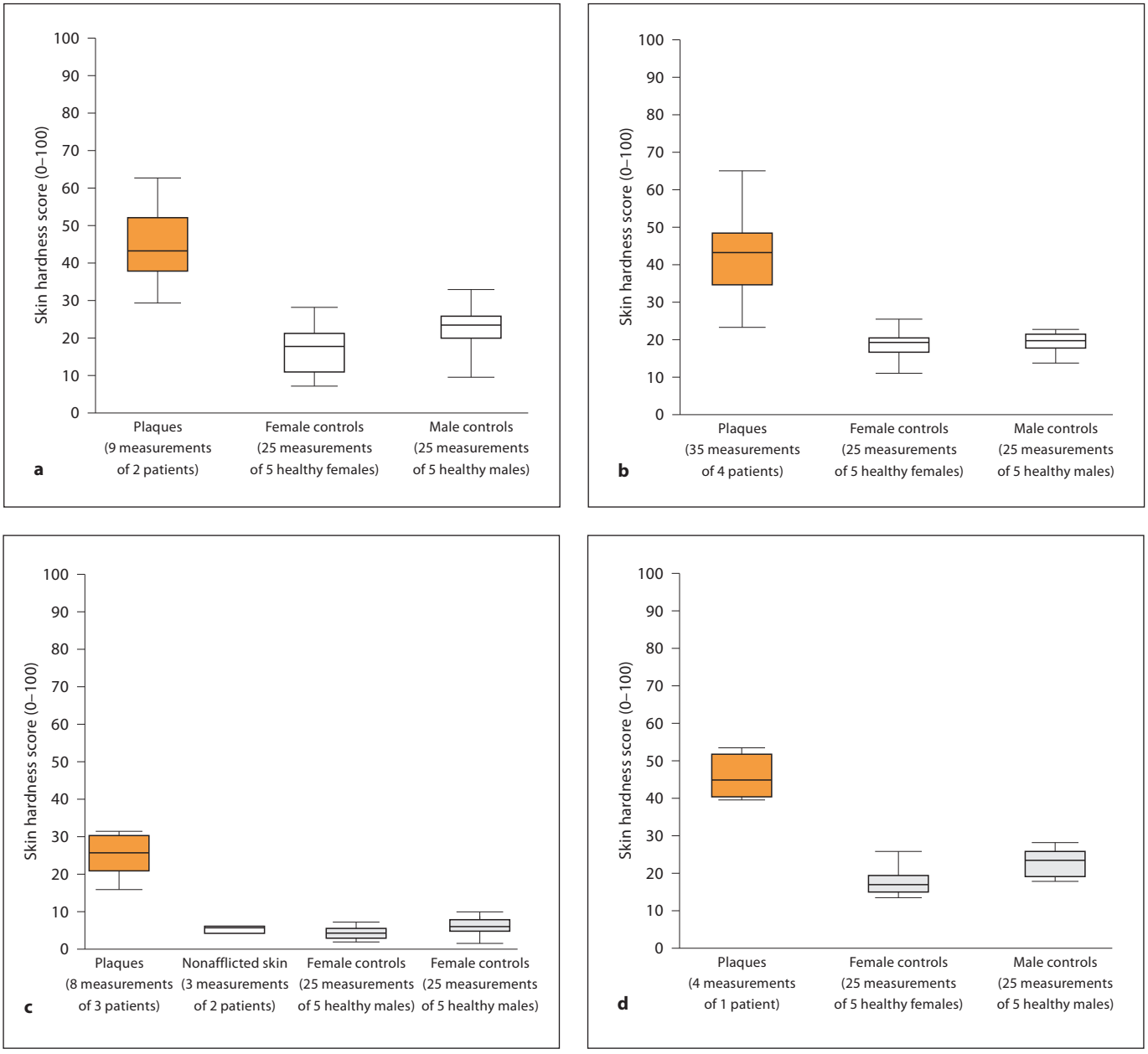
women was slightly softer in comparison to healthy men, probably due to the higher amount of subcutaneous fat tissue (fig. 6a, c, d).

In 3 patients (No. 8–10) with CS, the decrease in sclerosis over the course of wIRA(+VIS) treatment was documented using durometry. The durometer measurements could be easily performed and were perceived as being painless and found to be helpful in the documentation of the severity of sclerosis and in monitoring the therapeutic process. Thus, durometer monitoring contributes to compliance since subjective clinical improvement was reflected in objectively improved durometer values.

One female patient who had had GCS (No. 8) with tendinitis and movement restriction and chronic pain of the forearm for 1 year improved during the first weeks of treatment (fig. 5), but showed rapid deterioration during a 5-week vacation without irradiation. Once the wIRA(+VIS) treatment recommenced, the skin status improved (fig. 5), but deteriorated again after cessation of the irradiation, indicating an efficacy of wIRA(+VIS) irradiation in this case for the duration of its use.

Discussion

In the current study, wIRA(+VIS) irradiation led to a marked improvement in 7 out of 10 patients. In another patient (No. 8), a mild to moderate improvement was documented, but she developed a worsening after cessation



Color version available online

Fig. 6. a Durometry measurements of the skin hardness of the back of the hands (**a**), of the forearm (**b**), of the abdomen (**c**) and of the thigh (**d**).

of irradiation. In 2 patients with GCS (No. 2, 10), in which UVA1 had previously failed, the applied wIRA(+VIS) regimen (with limited irradiation frequency, irradiation time and treatment period) resulted in only a slight decrease in sclerosis. It is unlikely that the impressive changes observed in 8 patients were due to spontaneous remission since: (1) the disease activity had persisted for years before treatment in all patients, (2) the tendency for spontaneous remission of established sclerosis in LCS and GCS was lower in comparison to early, superficial inflammatory cases [1], (3) 1 patient showed an improvement when she was irradiated 3–5 times a week but showed deterioration twice after cessation of treatment. Our data are in line with a case report on successful wIRA(+VIS) treatment of a long-standing ulcerating variant of CS on the lower leg [8].

The pathogenetic mechanisms of CS are as yet unknown, but it is characterized histologically by inflammation and fibrosis/sclerosis [1, 14]. To explain wIRA(+VIS) effects in CS, an influence on angiogenesis, immune mechanisms and/or collagen metabolism should be considered.

Structural cutaneous vascular abnormalities with dysfunctional perfusion and decreased skin temperature have been observed in patients with systemic sclerosis [14]. Thermal imaging showed the skin temperatures of patients with systemic sclerosis to be approximately 28.0°C (baseline), compared to 34.5°C in healthy controls [14]. In contrast to systemic sclerosis, no evidence has been presented thus far for vasculopathy in CS. Considering the potential beneficial effects of wIRA(+VIS) in systemic sclerosis, it is important to note that the wIRA(+VIS) device used does not emit infrared C and almost no infrared B, and has relatively low water absorption bands within infrared A. Therefore, overheating effects due to infrared radiation are avoided. During wIRA(+VIS) application in vivo, Mercer and de Weerd [15] observed a rise in the mean skin temperature from 32.5 to 38.2°C. An increase in tissue temperature by wIRA(+VIS), without overheating of the skin, with improved tissue perfusion, oxygenation and metabolism [9, 11, 15] might therefore contribute to the improvement of the disease condition.

Elevated collagen metabolism with increased collagen degradation due to increased matrix metalloproteinase 1, cross-link pyridoline and deoxypyridinoline as well as increased collagenase mRNA levels can be observed after UVA1 irradiation [4]. UVA1 is known to be an effective therapeutic option in CS [4]. It has been claimed that infrared radiation is capable of modulating fibroblast function through an induction of heat shock proteins (e.g. heat shock protein 72) or by an upregulation of matrix metalloproteinases which could influence collagen degradation [16]. However, a recent analysis of these and other studies in which the effects of infrared radiation were investigated showed that in many cases – due to the experimental setup used – it was not possible to differentiate between the direct effects of infrared irradiation and secondary temperature effects, especially when inappropriately high irradiances were used without any regulation of temperature to physiological values in cell culture dishes [17]. It should be noted that in appropriately temperature-controlled (37°C) in vitro experiments, wIRA(+VIS) – in contrast to heat or UVA1 [4, 18] – did not induce the expression of MMP-1 [19, 20]. These data show that wIRA(+VIS) irradiations under controlled temperature conditions (37–39°C), as applied in clinical practice [9–11, 15], have no currently known influence on this aspect of collagen metabolism [19, 20].

In CS perivascular inflammatory cells and release of profibrotic cytokines have been observed [1]. Irradiation with wIRA(+VIS) can have an anti-inflammatory and immunomodulatory effect [11, 13]. Immunomodulation following wIRA(+VIS) irradiation has already been described after treatment of verrucae vulgares [13]. While the mechanisms underlying such an immunomodulation have not yet been fully identified, increases in tissue temperature and tissue oxygen partial pressure and nonthermal direct effects on cells could play a role [11, 13]. Moreover, PDT has been reported to result in an improvement of CS [21]. The interaction of the VIS component with endogenous protoporphyrin IX (and/or protoporphyrin IX produced by physiological skin bacteria) might thus act as a mild form of PDT [10], modulating immune responses in scleroderma plaques.

In earlier investigations durometry has been proven to be reliable in the assessment of morphea [23–25]. Moreover, Kissin et al. demonstrated that durometry enables detection of even minor changes in skin hardness over time [24]. For our investigations we used a new durometer and observed – in line with previous studies – that durometry is painless and can be easily performed [23–25]. Durometry was suitable for the documentation of the success of therapy and to motivate patients to continue treatments.

In conclusion, a decrease in sclerosis in all patients has been demonstrated in this study with a long-lasting marked improvement in 7 out of the 10 patients of the study cohort. One advantage of the treatment is its easy use, which would make therapy at home feasible. To date, there are no known side effects. wIRA(+VIS) therefore appears to be a promising new treatment modality for CS that warrants further evaluation in larger controlled studies.

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